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Mary S. Wolfe, PhD,
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November 28, 2000

Dear Dr Wolfe,

As promised in my recent letter, I attach my submission on talc for consideration by NTP. I look forward to hearing from you concerning the scheduling of my short oral presentation to address the salient points in my submission.

Yours Sincerely,

Graham W Gibbs
MSc PhD ~~LRSC~~ ROH.

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AN EVALUATION OF THE EPIDEMIOLOGICAL EVIDENCE CONCERNING "TALC" AND RESPIRATORY CANCER IN HUMANS WITH SPECIFIC ATTENTION TO "TALC" AS PRODUCED BY THE GOUVERNEUR TALC COMPANY [A SUBSIDIARY OF THE R.T. VANDERBILT COMPANY INC] AT ITS MINES IN NEW YORK STATE.

GRAHAM WILLIAM GIBBS MSc PhD LRSC ROH

Safety Health Environment International Consultants Corp.,
Alberta, Canada.

November 27, 2000

AN EVALUATION OF THE EPIDEMIOLOGICAL EVIDENCE CONCERNING "TALC" AND MALIGNANCY IN HUMANS WITH SPECIFIC ATTENTION TO "TALC" AS PRODUCED BY THE GOUVERNEUR TALC COMPANY [A SUBSIDIARY OF THE R.T. VANDERBILT COMPANY INC] AT ITS MINES IN NEW YORK STATE.

GRAHAM WILLIAM GIBBS MSc PhD LRSC ROH

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and
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QUALIFICATIONS AND EXPERIENCE.

I received a Baccalaureate [BSc] degree in Geology and Chemistry at the University of London, England in 1966, a Master of Science [MSc] degree in Geological Sciences [Dean's Honours List] at McGill University in 1969 and a Doctor of Philosophy degree [PhD] in Epidemiology [Dean's honours List] at McGill University, Montreal, Canada in 1972. I am a Licentiate of the Royal Society of Chemistry [LRSC] and a Registered Occupational Hygienist [ROH] through the Canadian Registration Board of Occupational Hygienists. I have worked in the occupational and environmental health fields for more than 40 years. My curriculum vitae and list of publications are attached [APPENDIX A].

BACKGROUND.

The R.T. Vanderbilt Company Inc., through the Environmental Sciences Laboratory, Brooklyn College of the City University of New York, have requested that I undertake a review of the various epidemiological studies of workers employed by the Gouverneur Talc Company in New York State to determine if they support or otherwise the designation of talc from this deposit as a carcinogen. The following is my report on this matter.

DEFINITIONS

The "Draft Background Document" for "Talc Asbestiform and Non-Asbestiform" is not clear in its definition of "Talc Asbestiform". The summary statement on page iii and v refer to: "Talc containing asbestiform fibers". Does this mean talc containing asbestos fibres or talc containing non-asbestos asbestiform fibres, both or talc containing elongated particles with aspect ratios exceeding 3:1, but with diameters less than about 3-4 micrometres? On page 5, the document notes "Natural talc deposits and commercial talc products are found to contain serpentines (chrysotile, antigorite and lizardite) and fibrous and non-fibrous amphiboles [Rohl et al 1976]. This form is also known as asbestiform talc, talc [containing asbestos] or talc containing asbestiform fibres."

If this is the definition being adopted by NTP, and if the term fibrous amphiboles in this definition refers to amphibole asbestos, then, it is not clear why a separate nomination would be needed for talcs containing asbestos as the asbestos minerals have already been classified as carcinogenic. The presence of asbestos fibres in a talc does not render the mineral talc carcinogenic, but the mixture, dependant on the asbestos fibre type, fibre dimensions and percentage may increase cancer risk.

On the other hand, the "Draft Background Document" cites studies of GTC miners and millers in support of the nomination. Because of this, it must be assumed the author of the "Draft Background Document", RG1 and RG2 consider that these workers are exposed to "talc asbestiform". This presents a definition problem because there is expert mineralogical opinion that GTC workers are not exposed to asbestos although they may be exposed to cleavage fragments meeting the OSHA definition of a fiber, talc fibers and transitional fibers. I will leave this technical issue to those who have studied the ore, product and airborne dusts and in this submission, the term "GTC talc" will refer to the mixture of minerals produced as "talc" by the Gouverneur Talc Company from its New York Deposit and include all its components. When the unqualified term "talc" is used, it refers to the minerals and mineral habits present in a particular talc.

CRITERIA TO DECIDE WHETHER GTC TALC IS CARCINOGENIC

According to the background document, listing a substance as a "known human carcinogen" requires that there is sufficient evidence from studies in humans, "which indicates a <u>causal</u> relationship between exposure to the agent, substance or mixture and human cancer". The criteria for listing a substance as "Reasonably anticipated to be a human carcinogen" is "There is limited evidence of carcinogenicity from studies in humans, which indicates <u>causal interpretation is credible but that alternative explanations such as chance, bias or confounding factors could not adequately be excluded</u>" or there is sufficient evidence of carcinogenicity from studies in experimental animals.

The report of the Carcinogens review group RG1 concluded that talc containing asbestiform fibers is known to be a human carcinogen and RG2 concluded that talc containing asbestiform fibers is reasonably anticipated to be a human carcinogen. Both conclusions are based largely on studies of talc miners and millers and "asbestos" exposure is mentioned.

In order to correctly interpret the results of the GTC worker studies, it is important that any pre-conceived notion that asbestos may be present and must be responsible for any increased respiratory cancer risks be set aside. The evidence for or against classifying talc as produced by GTC as a carcinogen should rest on the evidence from the studies of talc workers and determine if the exposures are <u>causally</u> associated with increased risks of respiratory cancer or that a <u>causal</u> interpretation is credible. The experimental data are part of this evaluation. Experimental data will not be discussed in this report, but must be considered in relation to plausibility. Some of the criteria which are often used in deciding on causality in epidemiological studies are listed in APPENDIX B.

LUNG CANCER RISKS IN GTC WORKERS.

The first study to include some GTC workers was a proportional mortality ratio [PMR] study of miners and millers in New York State who had had 15 or more years of exposure to talc dust in 1940 or between 1940 and 1965 [Kleinfeld et al 1997]. Follow-up began in 1940 which was, seven years before GTC began production. It is evident that the number of GTC workers included in this cohort would have been very limited. The study indicated a high PMR from lung cancer, but this needs to be interpreted with considerable caution as 30% of deaths in the cohort were due to pneumoconiosis or complications, a cause of death not common in the referent US general population. It was also based on the US proportional mortality for only one year. The role of smoking was not assessed and the characterization of the dusts to which the workers were exposed was general with no reference to mineral habit. All cases of lung cancer had initial exposure before 1945 when wet drilling was introduced, but "there was no evidence to indicate that there was a direct relationship between duration of exposure prior to the onset of wet drilling and the occurrence of pulmonary carcinoma". In retrospect, this is perhaps the first indication that the lung cancer risk may not be exposure related.

Brown et al [1980] reported on the NIOSH study. This study has been well critiqued [eg: Gamble 1985] and there is little value in revisiting this cohort of 398 workers as the study has now been updated and superseded by more recent information. For the record, the study did not examine exposure-response or take smoking into consideration.

Stille & Tabershaw [1982] studied 744 men employed January 1 1948 through December 31 1977. After exclusions for lack of information, 655 white male talc workers were available for analysis. The mortality from lung cancer compared to US white males was not statistically significantly increased [SMR = 157 Observed = 10] in men who worked at the plant [assumed to be GTC] but was statistically significantly greater [SMR=214 observed 8] in men who worked elsewhere before joining the plant. Incidentally, most of the criticisms levelled at this report by IARC and noted the "Background Document, page 20", also apply to the original NIOSH study in 1980 and Vermont study by Selevan et al [1979] as none of these studies took account of smoking or involved an examination of exposure-response.

Lamm et al [1988] reported on what appears to be essentially the same cohort as studied by Stille and Tabershaw [1982]. They found that 425 had worked for GTC for more than one year and 280 for less than one year. They categorized each job on the pre-employment history by likelihood of increasing lung cancer risk. The overall mortality from respiratory cancer was elevated [SMR = 240], but as reported by Stille & Tabershaw [1982], the lung cancer mortality was concentrated in men employed for 1 year or less [SMR=317] and concentrated in those who had worked in jobs carrying a lung cancer risk before joining GTC [SMR=316]. The respiratory cancer risk was lower in persons with longer duration of employment. Importantly they noted that there were no differences in the initial jobs assignments at GTC for workers who left within 1 year and those who stayed. This observation does not support the hypothesis put forward by Brown et al [1980] that the excess lung cancer risk is due to short high exposures encountered by short-term workers. This study did not have smoking information available.

In 1990, Brown et al [1990] expanded the original NIOSH cohort definition, increasing the cohort size to 710 white males employed at any time between 1947 and 1978, and updated the vital status to December 1983. The overall SMR for lung cancer was still elevated [SMR 207] compared to the experience of US males. However, the authors found that the SMR for workers with 20 or more years of latency and less than 1 year tenure was 357 [CI: 154, 704], while those workers with more than 20 years of latency and more than 1 year of tenure had an SMR = 178 which was not statistically significant; ie could have occurred by chance. Again, detailed smoking histories were not available. This difference in risk between the short term and long term employees would not be the pattern anticipated if the lung cancer excess were related to the GTC exposures unless the short-term workers had higher talc exposures than longer term workers. The study by Lamm et al [1988] did not suggest that this was a likely scenario. Again an exposure response study was not undertaken. This report was not cited in the Background Document.

The reason for the excess lung cancer reported by Kleinfeld et al [1967] and Brown et al [1980] was not known in 1986 when IARC [1987] deliberated on talc. It was probably inferred because the studies involved talc miners and millers and minerals such as "tremolite and anthophyllite [asbestiform and nonasbestiform habits] were mentioned. In fact, there had been no exposure-response studies and smoking had not been taken into account. The results of the larger NIOSH study [Brown et al 1988] or the results of studies discussed in the following paragraphs were not available to them.

Four years after the IARC [1987] review of Talc, the interpretation of the data were still being debated [Morgan & Reger 1990]. However by that time, it was known that:

> There was an increased mortality from lung cancer in GTC cohort members. This was observed by all researchers, but this should not be surprising as they were studying the same or overlapping cohorts.
>
> The excess mortality from lung cancer was greater in the workers employed for less than 1 year than in those employed for more than 1 year.
>
> The excess did not seem to be due to different initial job assignments for workers with short and long term employment.
>
> The excess lung cancer mortality seemed to be explained in part by prior employment in other "cancer risk" industries.

Since 1990, two studies have become available which are extremely important in understanding the epidemiological studies and are the only ones available which provide information on which to determine whether or not the excess lung cancer in GTC workers is associated with exposure to GTC talc.

The first study is the nested case-control study reported by Gamble [1993]. The 22 cases selected for study were those dying with lung cancer in the NIOSH update cohort of 710 white males studied by Brown et al [1990]. There were 3 controls per case, matched as closely as possible for date of birth and date of hire. Controls had to survive the case. Work history information was obtained from GTC files and tobacco use and additional work history information was obtained from the cases and controls or from relatives and friends. Smoking status was obtained for all cases and controls. A panel of epidemiologists and occupational hygienists classified the non-talc jobs held by the cases and controls as to the risk of lung cancer associated with them on a scale of probable [score 3], possible [score 1], or none [score 0]. The composite score was developed for each man by multiplying the score for each job by the time spent in that job and summing the results over all jobs. The total scores were broken into 4 categories and estimates of the odds ratios for each category were then used to determine if this index of work at other than GTC jobs increased the risk of lung cancer. The author analyzed the data with and without non-GTC talc experience and took latency into account. The important findings were as follows:

- In an analysis to determine lung cancer risk in relation to smoking, the odds ratio in smokers was 5.71 when the odds ratio for ex-smokers and no-smokers was set at 1.00. This risk was 6.55 in persons smoking more than >40 cigarette/day smokers. There were no no-smoking lung cancer cases. It is evident that smoking has the potential to play an important role in the lung cancer experience of these workers.

- All workers had had non-GTC jobs. However, there was no increasing trend in the odds ratios for the risk of lung cancer with the "non-talc employment" indices. This indicates that workers were not dying of lung cancer as a result of working elsewhere. It is unfortunate that the author did not also carry out this analysis with a 20 year latency, to determine whether the most relevant employment in non-GTC jobs was associated with an increased lung cancer risk as this was suggested by previous research. For this reason the possibility that work elsewhere contributed to the lung cancer risk cannot be totally excluded.

- When only smokers were analyzed, the case control studies showed that the odds ratios for lung cancer risk by tenure at GTC with and without a 20 year latency showed no increasing trend and odds ratios remained below 1.00 as tenure increased. In fact the results consistently suggest that the risk of lung cancer decreased significantly with tenure at the plant. This pattern did not change in any important way when non-GTC talc exposure were added. This is not consistent with exposures at the plant being responsible for the apparently increased risk of lung cancer in the cohort unless tenure does not reflect exposure.

- The decreasing pattern of risk with increasing tenure would occur if the risk of the short tenure workers was elevated [for whatever reason]. The fact that it was increased was suggested in previous studies. In this regard it is important to note that Gamble did analyze the data excluding men with less than 20 years latency and less than both 1 year of tenure and men with less than 3 months of tenure. In the latter case, 11 lung cancer cases were removed. The case-control analysis restricting the analysis to smokers and setting the odds ratio for the 3 months - 5 years of employment at 1.0 showed that workers employed 15-34 years with more than 20 years since first employment had an odds ratio of 0.73.

The use of tenure as a surrogate for exposure has limitations. First, if there are non-exposed workers, tenure assumes exposure. Second, if there are large variations of exposure over time, tenure would not reflect these and this could affect the tenure-response relationship observed. Further the numbers become small [9 cases] when the short term workers are excluded. In spite of these limitations, the absence of an increasing trend of lung cancer risk with increasing tenure after a latency of 20 years and after eliminating short term workers is not supportive of a GTC employment etiology.

The question now remains as to whether the dust exposure of workers in the GTC mine and/or mill are associated in any way for the increased risk of lung cancer. The second study attempts to answer this question [Delzell et al 1995]. It is unfortunate that this study has not been published. However, it was reviewed by 4 reviewers and their collective comments are available [Boehleke 1994].

In this study, individual cumulative respirable dust exposures were estimated for all GTC cohort members. These estimates were based on a job-exposure matrix. This consisted of an average respirable dust concentration in each work area and calendar year for the period 1948 through 1989. Historical dust concentrations exposures in various work areas by time periods were rated by a knowledgable panel of GTC employees. Special dust sampling surveys were conducted and paired respirable dust and dust count samples collected and used to convert historical dust count data to gravimetric respirable dust concentrations.

Baseline dust concentrations were based on the results of the special survey and a NIOSH survey. Past dust concentrations were then estimated by weighting baseline concentrations by the scores developed for the various time periods. These estimated past concentrations were then validated against historical dust measurements. It appears that a carefully considered approach was used to obtain respirable GTC talc exposure estimates which could be used to develop individual exposures for use in evaluating exposure-response.

The cohort consisted of 818 white men who worked for at least 1 day at the GTC from 1948 through 1989 and who had known birth and employment dates. The follow-up period was January 1 1948 through December 31 1989. There were 46 men with no work history who had a median duration of employment of 0.19 year. Their exclusion would not impact the risks of longer term workers. Twenty eight percent of the cohort were deceased. Causes of death were available for 222 [98%] of the 225 deaths. It should be noted that 344 [42%] of workers worked for <1yr and 521 for <5 years.

Compared to US white men, the cohort had an SMR from all causes of 141 [95% CI=123-161]. Excess mortality was observed for several causes of death including circulatory diseases, non-malignant respiratory diseases and cancer [SMR = 154, 115-200, observed = 54]. The cancer excess was mainly due to lung cancer [SMR 254, 173-361 observed = 31]. This finding of an overall excess of lung cancer is similar to that of earlier investigators. The use of local rates did not change the results.

For lung cancer, 22 of the 31 deaths occurred in men with less than 5 years of employment. The SMR did not rise with increasing length of employment within any category of years since hire. However, a statistically significant excess was present for the group of workers with less than 5 yrs of employment and more than 20 years since hire. For workers with more than 5 years of employment with 20+ years since hire there was a non statistically significant increased risk of lung cancer [SMR = 215, 86-442, observed = 7]. Thus, in the 20+ years since hire workers, the SMR of those employed for a short period exceeded that of the longer term workers. This argues against a GTC work related factor being responsible for the observed increased risk of lung cancer.

The overall excess of lung cancer was concentrated in men employed in the underground mine [SMR = 440, 262-695, Observed = 18]. In fact the excess lung cancer mortality was in men who were only employed in the mine [SMR = 473, 280-747, Observed =18].

In contrast, there was only a small non-significant increase in lung cancer mortality in mill workers [SMR = 139, 56-287, observed = 7], a group with similar exposures to the underground workers. Such an increased risk might be explained by smoking [but this cannot be determined as smoking data were not available]. NMRD was in excess in millers [SMR = 321] and in underground miners [SMR = 349]. If talc were responsible for the excess lung cancer, one would have expected the same pattern of mortality of lung cancer mortality in both the millers as well as miners.

Lung cancer mortality was also increased among men who were <u>exclusively</u> employed in unexposed jobs [SMR= 443, 87-1264, Observed = 3]. This again, on small numbers, argues against a GTC talc etiology for the lung cancer excess.

When exposure-response was examined, there was an inverse relationship between lung cancer mortality and estimated cumulative dust exposure. The relative risk [RR] was 0.66 [CI: 0.32-1.4] for men with cumulative exposures greater than or equal to the median exposure versus those below the median value. Analyses by quartiles also suggested an inverse association. When men with less than one year of GTC employment were excluded, the RR for the same comparison was 0.62 [CI: 0.22-1.8].

All 7 subjects who had reportedly died with pneumoconiosis or interstitial lung disease had cumulative exposures above the cohort's median value. This suggests that the cumulative index of exposure is relating sensibly to mortality from pneumoconiosis, but that there is no evidence that the cumulative exposure to GTC talc relates sensibly to the lung cancer risk observed in this industry.

Two deaths from mesothelioma were reported. The one mesothelioma case had only 15 years between hire and death. In the Quebec chrysotile miners and millers there was not a single case with less than 20 years from first exposure to death. The other mesothelioma case had worked for several years on the construction of another talc mine before his GTC employment. At GTC he worked as a draftsman during mill construction in 1948-49 and worked outdoors. After leaving GTC he worked in removing, installing and maintaining oil heating systems where the possibility of asbestos exposure cannot be excluded. Thus, neither case is likely linked to GTC employment.

In addition to examining the relationship between cumulative respirable dust exposure and lung cancer mortality [not done in any other study], this cohort was larger than the original and updated NIOSH studies; the follow-up period was longer by 7 years than the most recent NIOSH study; analyses were performed using national, regional and local rates; internal comparisons were done and a major effort was undertaken to ensure that the cohort was complete using IRS 941 records. Unfortunately, tobacco consumption was not taken into account and is a weakness in hat we do not know whether the persons with low cumulative exposures smoked more than those with high cumulative exposures. I think this is unlikely, based on my experience with other industries, but we do not know.

While the authors note that the use of an inappropriate index of exposure is another potential weakness, it would reasonably be expected that a higher respirable dust exposure would mean a higher exposure to any pertinent carcinogenic constituent of the GTC talc if there were any, so while at most, reducing the slope of an exposure-response relationship, it would be highly unlikely to reverse it. It is unfortunate that Delzell et al did not gather smoking or non-GTC employment information and carry out a nested case-control study to determine if they offer a possible explanation for the decreasing risk of lung cancer with increasing cumulative GTC talc dust exposure.

INFORMATION FROM OTHER STUDIES

There are other studies, some of which were not evaluated by IARC which are pertinent to the issue of respiratory cancer risks associated with talc.

Rubino et al [1976] followed 1514 miners and 478 millers in Italy. They separated the miners and millers because they considered the mine air dust to include certain amounts of inhalable silica. The talc was reasonably well characterised with no amphibole or chrysotile asbestos detected "in any amount in rocks and in inclusions". Rubino et al examined the risk of lung cancer in relation to 3 categories of cumulative exposure levels and showed no increasing risk with either level of latency. They also did not find any increased risk in miners compared to millers. The study used an external comparison population in the area and also internal comparisons. IARC expressed concerns about their comparison group. In a later paper [Rubino et al 1979] expected deaths were recalculated using Italian white male rates, which would have eliminated this concern. There was still a deficit of lung cancer in the miners and millers and no increase in risk with increasing cumulative exposure to "talc". This study does not support an increased lung cancer risk associated with their talc which contained quartz, muscovite, chlorite, garnet, carbonates [calcite and magnesite]. Talc or other fibers were not mentioned as present or absent.

Wergeland et al [1990] conducted a small study of 94 talc miners and 295 talc millers in Norway. Their talc was described as "non-asbestiform" talc with low quartz content. However, the talc contained trace amounts of tremolite and anthophyllite. Fibres were reported to have been detected near the "detection limit for optical microscopy" and low fiber content confirmed by electron microscopy. It is not known whether these were asbestos fibers or talc fibers. The main minerals in the talc deposit are talc and magnesite. In addition the ore contains magnetite, chromite, chlorite and antigorite with adjacent rocks containing serpentine, mica, feldspar, calcite and the amphiboles, hornblende and tremolite. Fibers, identified as tremolite, anthophyllite and talc were particles fulfilling the fiber definition of having a length: diameter ratio greater than 3:1. Smoking information was available. The numbers in the mine were too few to meaningfully interpret, but in the mill there was no excess incidence of lung cancer.

Selevan et al [1979] carried out a mortality study of what was described as "non-asbestiform" talc in Vermont. Quantitative estimates of "talc" exposure were not made, so the talc exposure-response relationships were not examined. It was of interest that there was a significant increase in respiratory cancer mortality in the miners but not in millers. It is perhaps relevant that if "talc" were responsible for the increased risk of lung cancer, then one would have expected to see the excess in both the millers and miners.

COMMENT

There seems to be little doubt that the overall lung cancer risk in the various GTC cohorts is elevated. On the other hand, virtually all the epidemiological evidence points away from the lung cancer increase being related to the GTC talc exposure. The excess lung cancer in the GTC cohort is present in miners but not in millers. Tenure and cumulative exposure, trend in a direction contrary to that expected if there were a link with GTC talc exposure. This trend holds when short term workers are excluded. One can only speculate on reasons for the high overall mortality and mortality from lung cancer. Smoking seems one likely candidate, but seems unlikely to explain some of the very high SMR's observed for underground miners. Miners encounter minerals which may entail exposures which are diluted with other dusts in the mill, so the exposure of miners is probably different from millers qualitatively. One possibility which has not been evaluated is whether workers were migrants. If this were the case, neither US or local rates would be appropriate and might provide spuriously increased SMRs.

CONCLUSION

1. NTP needs to carefully define what is meant by "Talc Asbestiform".

2. The reason for the overall excess lung cancer in cohorts of GTC workers is still not known. However, it is clear that a statistically significant excess of lung cancer is present in underground miners but not in millers. The lung cancer risk does not increase with increasing tenure or cumulative exposure to respirable GTC talc dust.

3. The evidence does not establish a link between GTC talc exposure and mesothelioma.

4. Collectively the currently available epidemiological studies of GTC workers do not support a causal relationship between GTC talc and respiratory cancer.

5. The currently available epidemiological studies of GTC workers do not support the premise that a causal relationship between GTC and respiratory cancer is credible.

6. In the absence of firm human data establishing a link between GTC talc exposure and respiratory cancer, biologic plausibility depends on an evaluation of the experimental data relating to GTC talc and its constituents. This has not been evaluated in this report.

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APPENDIX A

Rev. 10.00

CURRICULUM VITAE

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FAX NUMBER:	403 987 4901

DATE OF BIRTH:	February 12 1941
BIRTHPLACE:	Cinderford, Gloucestershire, England
CITIZENSHIP:	Canadian and British
MARITAL STATUS:	Married with one son.
LANGUAGES:	English (mother tongue); French (very good)

EDUCATION

SCHOOLS

Primary	- Bilson School, Cinderford, England.	1946-1951
Secondary	- Monmouth School, Wales.	1951-1959
University	- Sir John Cass College, University of London, London, England.	1960-1966
	- McGill University, Montreal, Canada.	1967-1972

GENERAL CERTIFICATES OF EDUCATION

Ordinary Level:	- Latin, French, Greek, Physics, Elementary Mathematics Additional Mathematics, English Language, English Literature, Divinity
Alternative Ordinary:	- Chemistry, General Mathematics, Physics, Economics
Advanced Level:	- Chemistry, Physics, Mathematics for Science.

UNIVERSITY DEGREES

1966	B.Sc.(London) Geology and Chemistry
1969	M.Sc.(McGill) Geological Sciences (Dean's Honours List)
1972	Ph.D.(McGill) Epidemiology & Medical Statistics (Dean's Honours List)

PROFESSIONAL QUALIFICATIONS

LRSC	Licentiate of the Royal Society of Chemistry
ROH	Registered Occupational Hygienist - Canadian Registration Board of Occupational Hygienists

MAIN APPOINTMENTS

1988 -		Safety Health Environment International Consultants Corp..
		President
1985 - 1990		Government of Alberta
	(1985-1987)	Executive Director, Occupational Health Services, Occupational Health & Safety, Division.
	(1987-1988)	Executive Director, Occupational Health Services, Community & Occupational Health.
	(1988)	Executive Director, Industry & Technical Services, Community & Occupational Health
	(1988)	Acting Managing Director, Occupational Health & Safety Division.
	(1988 -1990)	Executive Director, Policy & Professional Services, Alberta Occupational Health & Safety.
1980 - 1985		Celanese Canada Inc. Montreal, Quebec, Canada
	(1980-1984)	Director of Health & Safety Affairs
	(1984-1985)	Director, Health, Hygiene, R & D.
1966 - 1985		McGill University, Montreal, Quebec.
	(1966-1972)	Lecturer and Director, Environmental Laboratories, Department of Epidemiology & Health, Faculty of Medicine.

	(1972-1975)	Assistant Professor, Department of Epidemiology & Health.
	(1975-1980)	Associate Professor, Department of Epidemiology & Health.
	(1975-1985)	Associate Professor, Department of Mining & Metallurgy, Faculty of Engineering
	(1974-1980)	Founding Director, Institute of Occupational Health and Safety, (now Department of Occupational Health), McGill University
1959-1966		British Medical Research Council, London, England
	(1959-1963)	Technical Officer, Department for Research in Industrial Medicine, London Hospital, London, England
	(1963-1966)	Technical Officer, Air Pollution Research Unit, St Bartholomew's Hospital Medical College, London, England.

OTHER APPOINTMENTS

1986 -		University of Alberta, Edmonton Adjunct Professor, Department of Public Health Sciences.
1986 - 1996		University of Calgary, Calgary. Adjunct Associate Professor, Department of Community Health Sciences.
1980-1985		McGill University, Montreal, Quebec.
	(1980-1984)	Auxilliary Professor, Department of Epidemiology & Health
	(1980-1985)	Auxilliary Professor, School of Occupational Health
1961-1963		East London College of Commerce, London County Council
	(1961-1963)	Part-time Instructor in Mathematics

MEMBERSHIPS IN PROFESSIONAL ASSOCIATIONS

International Commission on Occupational Health (ICOH)
British Occupational Health Association (BOHS)
American Conference of Governmental Industrial Hygienists (ACGIH)
American Industrial Hygiene Association (AIHA)
Royal Society of Chemistry (RSC)
Canadian Occupational Health Association (COHA)
Alberta Occupational Health Society (AOHS)
Association québécoise pour hygiene, la sante et la securite du travail (AQHSST)

AWARDS

Recipient of Safety Equipment Australia Award to deliver Plenary Lecture at Australian Insititute of Occupational Hygiene Conference, Perth 1990.

COMMITTEES & OTHER ACTIVITIES

CURRENT (2000)

Member, International Board of Editors, Annals of Occupational Hygiene (Since 1992).

Temporary Advisor, World Health Organization International Program on Chemical Safety (IPCS) - Chemical Safety Card Program. (Since 1989)

Member, Scientific Committee on Fibres (previously Mineral Fibres) of the International Commission on Occupational Health.

Reviewer of grant applications for various agencies. (Since 1967).

Reviewer of manuscripts for publication in various scientific journals including the Annals of Occupational Hygiene, American Journal of Industrial Medicine, Scandinavian Journal of Work Environment Health, International Journal of Occupational and Environmental Health.

PAST

Member of Scientific Organizing Committee. An international workshop on Simian Virus 40 (SV40), asbestos and mesothelioma. November 9-10 1997.

Member of Canadian delegation to meet with British Health & Safety Executive to review health risks associated with chrysotile - Organized at request of Canadian and British Prime Ministers September 30 1997

Member of International Organizing and Scientific Organizing Committee - Workshop on the Health Effects of Chrysotile Asbestos, Contribution of Science to Risk Management Decisions, Montreal, September 15-17 1997.

Served as "Appointed Expert to the Appeal Board", Hazardous Material Information Review Act (May 1985 - June 1996; May 1997 - June 1997).

Invited Consultant, European Commission, Risk Management Workshop (January 1997).

Visiting Professor - University of West Indes (March 1997)

Joint organizer with Mr Thomas Schneider (Denmark), Workshop on full-scale test of fibre release during the use of MMMF products, Copenhagen Denmark (1996).

Member of the Editorial Board, International Journal of Occupational and Environmental Health (1994-1996).

Chairman, Scientific Committee on Fibres (previously Mineral Fibres) of the International Commission on Occupational Health (1989 - 1996).

Elected Member, Board of Directors, International Commission on Occupational Health (ICOH) (September 1993 - September 1996).

Coordinator of rapporteurs and editor of workshop report, Workshop on Man Made Fibres, Paris, February 2-5 1994.

Organizer, Workshop on the health risks associated with chrysotile asbestos, St Helier, Jersey, Channel Islands, November 14-17 1993.

Overseas Editor, Annals of Occupational Hygiene (Since 1976).

Member of the Expert Advisory Panel, Canadian Network of Toxicology Centres. March 15-17 1992.

Temporary Advisor, World Health Organization Regional Office for Europe & Chairman, WHO Consultation On Man-Made Fibres: Validity Of Methods For Assessment Of Carcinogenicity Of Fibres, Copenhagen, 19-20 May 1992.

Member, Scientific Committee "Workshop on indoor air quality and health" re: 1994 NATO workshop, Albuquerque, New Mexico. (Cancelled).

Co-Chairman of the Organizing Committee for the International Symposium on the Health effects of Low Exposure to Fibrous Materials, Nov 26-27 1991, Japan.

Invited participant, Workshop on "Approaches To Evaluating Toxicity and Carcinogenicity of Man-Made Fibers" CIIT, NC. November 11-13 1991.

Technical Resource to the Alberta (Industry, Labour and Government) Forum For Action on Occupational Health & Safety. (1989-91).

Member of Occupational Safety and Health Steering Committee on Biotechnology (Canada). (1989-90).

Member of Scientific Programme Committee, 23rd International Congress on Occupational Health, Montreal 22-28 Sept. 1990.

Organizer and Chairman of Mini-Symposium "Mineral Fibres - What of the Future?", 23rd International Congress on Occupational Health, Montreal, Quebec, September 26 1990.

Temporary Advisor to the World Health Organization Working Group on Indoor Air Quality: Inorganic fibres and other particulate matter, Kingston, Ontario 24-28 July 1990.

Co-Chairman of the Western Region Workshop on Human Health and Environmental Impact Assessment (sponsored by CAERC, FEARO, Environment Canada, H&W Canada, CPHA), May 8 - 9 1989.

Member,Federal- Provincial Advisory Committee on Occupational & Environmental Health (1985 - 1990).

Member, Committee on Bylaws/Ordinances of the Canadian Registration Board of Occupational Hygienists (1989-91).

President, President Elect, Past President and Member of Executive, Canadian Occupational Health Association (1986-89).

Member Technology & Research Advisory Committee, Alberta Government (1987-1990).

Member, Joint Consultative Committee (providing for coordination of activities of the Canadian Council of Resource & Environment Ministers (CCREM) and Federal Provincial Advisory Committee on Environmental and Occupational Health (1987-1990).

Scientific Secretary Committee on Mineral Fibres of the International Commission on Occupational Health (1988-1989).

Member, Alberta Chamber of Resource Industries' Task Force on Occupational Safety (1986-1989).

Co-Chairman, Health Committee, Acid Deposition Research Program (1986-1989).

Co-Chairman, Scientific Program Committee, Conference on Current Issues in Occupational Health, Edmonton Nov 12-13 1987.

Chairman, Scientific & Program Committee for the Canadian Occupational Health Association Conference "Occupational Health Services in Canada Through the Year 2000" held in Calgary, November 1986.

Member of the Scientific Committee for the International Commission Conference on Occupational Health Education (1985-1986).

Member of Working Group on the Pooling of Epidemiological Data,International Commission on Occupational Health (1984 -1987).

Member of Committee on Fibres of the International Commission on Occupational Health (Since 1984).

Member of the "Expert committee",International Agency for Cancer Research, Monograph on Aluminium Reduction,Iron and Steel Foundries,Coal Gasification and Coke production. (1983-84)

Member of Scientific Committee for Medichem Conference, Calgary 1983. (1983).

Member of Executive Board, Canadian Centre For Occupational Health & Safety (1982 -1985).

Governor, Canadian Centre for Occupational Health and Safety. (1982 -1985).

Member, National Research Council Subcommittee on Air of the NRC Committee on Scientific Criteria for Environmental Quality. (1982 -1985).

Member, Occupational Health Committee of the Formaldehyde Council. (1982-1985).

Member, Subcommittee on Risk Estimates,Advisory Committee on Radiological Protection, Atomic Energy Control Board (1981-1988).

Member of the Board and Executive Board, Conseil Quebecqois pour la Sante et Securite des Industries Textiles Primaire (CQSSITP). (1981 -85).

Member, Scientific committee of the Institut de Recherche et de Developpement de l'amiante (IRDA). (1980 -1985).

Member of Can/Asb SG/EG committee on Asbestos Fibre Definition and Fibre measurement. (1979-80).

Member, POGF Biomedical Group, National Academy of Sciences, Washington, USA (1979-1981).

Member, Comite des Fiches Techniques, Association pour L'Hygiene Industrielle du Quebec. (1979).

Consultant, Task Group on naturally occurring inorganic fibres, ASTM Committee E-34, (1978-80).

Chairman, Program Committee,Association pour l' Hygiene Industrielle au Quebec (1978).

Membre, Comite de Normalisation des Masques Respiratoires,Bureau de Normalisation du Quebec. (1978)

Member COPE Task Group on Respiratory Protection (1978).

Member of Committee on the Feasibility of Environmental Manpower Studies, Canadian Public Health Association (1976).

Member of Subcommittee 40, Descriptive/Comparative Methods, National Health Research and Development Program, National Health & Welfare Canada (1976 -1979).

Chairman and Organizer,Session, "Occupational Health",25th Annual Canadian Chemical Engineering Conference, Montreal (1975).

Member, Advisory Council, Centre for Resource Studies, Queens University, Kingston, Ontario. (1975 -1976).

Member of Task Force, Government of Quebec,"Orientation generale pour la Securite et la Sante en Milieu de Travail au Quebec" (1975 -1976).

Membre, Comite Interministeriel d'Hygiene et de Securite au Travail A.A.1489 -74 (1974).

Air Pollution Control Committee of the American Industrial Hygiene Association (APECC). (1974).

Session Arranger, American Industrial Hygiene Association Conference Program Committee (1973).

Member, National Research Council Committee on Scientific Criteria for Environmental Quality, Panel on Asbestos.

Member, ACGIH Committee on Aerosol Hazards (1972 -1974).

Secretary, Physics and Chemistry Panel of the Advisory Subcommittee on Asbestos Cancers to the Director of the International Agency for Research on Cancer, Lyon, France (1972).

Member of Working Committee on Physics and Chemistry of the Subcommittee on Asbestosis of the Permanent Commission on Occupational Health (1970).

Past member of various professional associations - Canadian Public Health Association; Alberta Public Health Association; Canadian Mineralogical Association.

PUBLICATIONS

A list of scientific publications is available on request.

Rev 2000.08

PUBLICATIONS

A. Books and Monographs

1.	McDonald JC	Gibbs GW, Manfreda J, White FMM. Storage as a Factor in Disease due to Mineral Dust. In:Multiple Factors in the Causation of Environmentally Induced Disease. Eds. HK Lee and P Kotin, New York 1972, Academic Press pp 185-201.
2.	Gibbs GW	Pintus P. Health and Safety in the Canadian Mining Industry. Centre for Resource Studies, Queens University 1978, 249 pp
3a.	Gibbs GW	Arhirii MI, Pierce RC. Effects of Particles on Human Health: Influence of Particle Size and Shape. National Research Council, Associate Committee on Scientific Criteria for Environmental Quality. NRC No. 18564, Ottawa 1982, 177pp
3b.	Gibbs GW	Arhirii MI, Pierce RC. Effets sur la Sante des Particules Absorbees Par Voie Respiratoire: Influence de Leur Taile et de Leur Forme. Comite Associe Sur Les Criteres Scientifiques Concernant l'Etat de L'environment. Conseil National De Recherches Canada. CNRC No 18565. 1982, 195pp.
4.	IARC	Working Group on the Evaluation of the Carcinogenic Risk of Chemicals to Humans. Polynuclear Aromatic Compounds, Part 3, Industrial Exposures in Aluminium Production, Coal Gasification, Coke Production and Iron and Steel Founding.IARC Monograph Vol. 34, International Agency for Research on Cancer, Lyon, France 1984, 219pp.
5.	Gibbs GW	Markham J (Eds). Occupational Health Services in Canada through the year 2000. University Press of Canada, Toronto, 1988, 241pp
6.	Gibbs GW	Occurrence, Production, Properties and Use of Naturally Occurring Mineral Fibres Other than Asbestos. In: Mineral Fibers and Health (Liddell D and Miller K Eds). CRC Press, Boca Raton, Florida 1991 pp 27-35

7. Gibbs GW — Kido M, Dunnigan J, Higashi T eds: Health Risks from Exposure to Mineral Fibres. North York Ontario. Captus University Publications. 1993 380 pp

B. Theses:

1. Gibbs GW — The Organic Geochemistry of Chrysotile Asbestos, Especially from the Eastern Townships, Quebec, M.Sc. Thesis, McGill University.

2. Gibbs GW — The Epidemiology of Pleural Calcification. PhD Thesis, McGill University, Montreal.

C. Scientific Journals and Proceedings

1969

1. Commins BT — Gibbs GW. Contaminating Organic Material in Asbestos. Brit. J. Cancer (23):358-362.

2. Gibbs GW — Some Problems Associated with the Storage of Asbestos in Polyethylene Bags. Amer Ind. Hyg. Assoc. J. (30):458-464.

1970

3. Gibbs GW — Collection and Storage of Samples in Organic Geochemistry. Geochim Cosmochim Acta (34):629-630.

4. Gibbs GW — Asbestos Fibre Contamination by Storage in Polyethylene Bags. In: Proc. Int. Conf. Pneumoconiosis, Johannesburg, 1969 Ed:HA Shapiro, Capetown (1970) Oxford University Press pp 165-167.

1971

5. Gibbs GW — Qualitative Aspects of Dust Exposure in the Quebec Asbestos Mining and Milling Industry. In: Inhaled Particles III, II Ed.WH Walton, Surrey, England (1971), Unwin Brothers Ltd., 783-799.

6. Gibbs GW — Hui HY. The Organic Content of Canadian Chrysotile. Amer Ind.Hyg. Assoc. J (32):519-528.

7. McDonald JC — McDonald AD, Gibbs GW, Siemiatycki J, Rossiter CE. Mortality in the Chrysotile Mines and Mills of Quebec. Arch Environ Health (22):677-685.

8. Jodoin G — Gibbs GW, Macklem PT, McDonald JC, Becklake MR. Early Effects of Asbestos Exposure on Lung Function. Amer. Rev. Resp. Dis. (104):525-535.

9. Gibbs GW — The Organic Geochemistry of Chrysotile Asbestos from the Eastern Townships, Quebec. Geochim Cosmochim Acta (35): 485-502

10. Cralley LJ — Ayer HE, Occella E, Holmes S, Gibbs GW, Amoudru C, DuToit RSJ. Evaluation of Asbestos Exposure in the Working Environment. Recommendation of Subcommittee on Asbestosis of the Permanent Commission and International Association on Occupational Health. Work Environment Health (8):71-73.

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1972

12. Gibbs GW — Comments on Standard International Methods for Evaluation of Asbestos Dust Exposure. In: Proceedings of the Subcommittee and International Colloquium on Asbestosis of the Permanent Commission and International Association on Occupational Health, September 1970, Sardinia, Italy, Eds B Pernis and PV Pelnar, Montreal, 1972.

13. Gibbs GW — Lachance M. Dust Exposure in the Chrysotile Mines and mills of Quebec. Arch Environ. Health (24): 189-197.

14. AIHA-ACGIH — Aerosol Hazards Evaluation Committee Statement. Amer. Indus. Hyg. Assoc. J (33):510.

15.	Cralley LJ	Ayer HE, Amoudru C, Gibbs GW, Holmes S, Ocella E, DuToit RSJ. Valutazione Dell Exposizione all Amianto negli Ambienti di Lavoro (Evaluation of Asbestos in Working Environment) Medicina del Lavoro (63) (1-2):1-6.
16.	Cralley LJ	Ayer HE, Amoudru C, Gibbs GW, Holmes S, Ocella E, DuToit RSJ. Evaluation of Asbestos exposures in the Working Environment. Industrial Medicine (41):28-30

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17.	McDonald JC	Becklake MR, Gibbs GW, McDonald AD, Rossiter CE. Epidemiological and Clinical Studies of Asbestos Workers in Quebec. In: Transactions of the Thirty Fourth Annual Meeting of the American Conference of Governmental Industrial Hygienists, San Francisco, California, May 14-19, 1972, p. 125.
18.	Gibbs GW	Allard D, Gray R, Corsillo A. Carbon Monoxide and Nitrogen Dioxide Exposure of Montreal Longshoremen. Can J. Pub Health (64):147-153.
19.	Gibbs GW	Hui H. A Pilot Investigation of Noise Hazard in Recording Studios. Ann Occup Hyg (16):321-327.
20.	Report of the	Advisory Committee on Asbestos Cancers to the Director of the International Agency for Research on Cancer In:Biological Effects of Asbestos. Eds. JC Gilson, V Timbrell and JC Wagner, Lyon 1973, IARC Scientific Publications No 8, pp 341-346.
21.	Gibbs GW	DuToit RSJ. Environmental Data in Mining. In:Biological Effects of Asbestos. Eds JC Gilson, V Timbrell and JC Wagner, Lyon 1973, IARC Scientific Publications No 8 pp 138-144.

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22.	McDonald JC	Becklake MR, Gibbs GW, McDonald AD, Rossiter CE. The Health of Chrysotile Asbestos Mine and Mill Workers of Quebec. Arch Environ Health (28):61-68.
23.	Gibbs GW	Lachance M. Dust-fibre Relationships in Quebec Chrysotile Industry. Arch Environ Health (28):69-71.

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24.	Joint	AIHA-ACGIH Aerosol Hazards Evaluation Committee. Recommended Procedures for Sampling and Counting Asbestos Fibres. Amer Ind Hyg Assoc J (36):83-90.
25.	Joint	AIHA-ACGIH Aerosol Hazards Evaluation Committee. Background Documentation on Evaluation of Occupational exposures to Airborne Asbestos. Amer Ind Hyg Assoc J (369:91-103.
26.	Gibbs GW	Fibre Release from Asbestos Garments. Ann Occup Hyg (19):143-149.
27.	Gibbs GW	Hwang CY, Physical Parameters of Airborne Asbestos Fibres in Various Work Environments - Preliminary Findings. Amer Ind Hyg Assoc J (36):459-466.
28.	Gibbs GW	Bogdanovic E. Trace Elements in Lung and Hair In: Recent Advances in the Assessment of the Health Effects of Environmental Pollution, Paris 1974, Office for Official Publications of the European Communities, Vol IV pp 2271-2283.

1977

29.	Gibbs GW	Baron P, Beckett ST, Dillen R, DuToit RSJ, Koponen M, Robock K. A Summary of Asbestos Fibre Counting in Seven Countries. Ann Occup Hyg (20):321-332.
30.	Gibbs GW	Results from Scientific and Practical Dust Measurements In: Proceedings of the International Colloquium on Dust Measuring Technique and Strategy, Warmensteinach, Germany, August 1977. Wirtschaftsverband Asbestzement E. V. Neuss, pp 92-98.

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32.	Gibbs GW	Health Hazards - Confrontation on the Job. A Perspective on the Issues and Solutions. Industrial Relations Centre 26th Annual Conference entitled "Are Health and Safety Negotiable?" Montreal McGill University, pp 66-78.
33.	Becklake MR	Soucie J, Gibbs GW, Ghezzo H. Respiratory Health Status of Children in three Quebec Urban Communities: An Epidemiologic Study. Bull Europ Physiopath Resp, (14):205-221.

1979

34.	Gibbs GW	Techniques of Asbestos Determination-Research Perspective. In: Mineralogical Association of Canada, Short Course in Mineralogical Techniques of Asbestos Determination, Quebec, May 1979, Ed. RL Ledoux. Mineralogical Association of Canada, Ottawa, pp 253-278.
35.	Gibbs GW	Techniques D'Evaluation De L'Amiante-Perspective De Recherche. In: Association Mineralogique Du Canada, Cours Intensif sur les Techniques de Determination Mineralogique de l'Amiante. Quebec, Mai 1979, Ed RL LeDoux. Association Mineralogique Du Canada, Ottawa, pp 281-308.
36.	Gibbs GW	Etiology of Pleural Calcification: A Study of Quebec Chrysotile Miners and Millers, Arch Environ Health (34) 76-83.
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1980

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45. McDonald JC — Gibbs GW, Liddell FDK. Chrysotile Fibre Concentration and Lung Cancer Mortality: A preliminary Report. In:Biological Effects of Mineral Fibers. Vol 1 Ed. JC Wagner, IARC Scientific Publications No 30, pp 811-817.

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1981

47. Gibbs GW — Identification and Quantification of Health and Safety Risks in Mining. CIM Bulletin No 74, pp 164-166.

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49. Hwang CY — Gibbs GW, The dimensions of Airborne Asbestos Fibres - 1. Crocidolite from Kuruman Area, Cape Province, South Africa. Ann Occup Hyg (24):23-41.

1982

50. Rowlands N — Gibbs GW, McDonald AD. Asbestos Fibres in the Lungs of Chrysotile Miners and Millers - A Preliminary Report. Ann Occup Hyg (26):411-415.

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53. ACRP — Risks of Low LET Radiation as Given in BEIR III and Previous Reports. ACRP-2. (Prepared by the Subcommittee on Risk Estimates, Muller J, Anderson TW, Gibbs GW, Myers DK, Newcombe HB, Elaguppillai V). Atomic Energy Control Board, Ottawa, 13pp

54. ACRP — Risk Estimates for Exposure to Alpha Emitters. ACRP-1. (Prepared by the Subcommittee on Risk Estimates, Muller J, Anderson TW, Gibbs GW, Myers DK, Newcombe HB, Elaguppillai V). Atomic Energy Control Board, Ottawa. 23pp

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1984

56. Gibbs GW — Mortality Experience in Eastern Canada. In:Health Protection in Primary Aluminium Production Vol 2 Ed. James P Hughes, London, International Primary Aluminium Institute, pp 59-69.

57.	Gibbs GW	Managing Workplace Health and Safety in the 1980s and Beyond. Accident Prevention. AIPA (31):2-5.
58.	Kobusch AB	Simard A, Feldstein M, Vauclair R, Gibbs GW, Bergeron F, Morissette N, Davis R. Pulmonary Cytology in Chrysotile Asbestos Workers. J Chronic Diseases (37) 599-607.
59a.	ACRP	Harm to the Offspring of Women of Childbearing age employed in the Nuclear Industry. ACRP-6. (Prepared by the Subcommittee on Risk Estimates, Muller J, Anderson TW, Gibbs GW, Myers DK, Newcombe HB, Hill GB, Avadhanula MR). Atomic Energy Control Board, Ottawa 10pp.
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60a.	CCRP	Assessment for Medical Legal Purposes of the Contribution of Occupational or Other Defined Exposure to Ionising Radiation as Causative Agent in Individuals Suffering From or Having Died of Cancer. ACRP-5. (Prepared by the Subcommittee on Risk Estimates, Muller J, Anderson TW, Gibbs GW, Myers DK, Newcombe HB, Hill GB, Avadhanula MR). Atomic Energy Control Board, Ottawa 4pp.
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1985

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1987

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1989

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1992

68. WHO — Validity Of Methods For Assessing The Carcinogenicity Of Man-Made Fibres. Executive Summary of a WHO Consultation 19-20 May 1992, Copenhagen. (G.W. Gibbs Chairman)

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74.	Gibbs GW	Browne K. & Valic F. (Eds) Health risks associated with chrysotile asbestos. A report on a workshop held in Jersey, Channel Islands, November 14-17 1993. Ann Ocup Hyg 38 399-426.
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APPENDIX B

FACTORS TO CONSIDER IN EVALUATING CAUSATION

Scientists and others are often faced with the difficult decision as to whether the available evidence is adequate to conclude that agent A causes disease B. There are two key elements to this problem that need to be considered. These are epidemiological and toxicological.

EPIDEMIOLOGICAL CRITERIA: There are criteria which are generally accepted by epidemiologists as being important in assessing the evidence for causality. The main parameters influencing studies are listed below...

BIAS

Normally, cohort studies are less subject to bias than case-control studies. However, there are circumstances where biases arise. A few of these are listed below:

<u>Cohort member selection bias</u>: If the records identifying persons to be included in a cohort are incomplete, bias may occur. Convincing information is needed to show that the omission of persons from the cohort does not lead to an over or under representation of persons who will get the disease or that the exposure of these omitted persons would not affect in any meaningful way exposure-response relationships.

An in-complete cohort or bias in the selection of cohort members detracts from the reliability of a study in terms of assessing causality.

Delzell made a special effort to ensure the completeness of her cohort.

<u>Diagnostic bias - non-cases</u>: If non-cases are included as cases (ie: contribute to the recorded number of observed cases), it is inevitable that any estimate of the number of expected cases based solely on validated cases will lead to a higher ratio of the observed number to the expected number of cases than would be found in the absence of this bias.

The inclusion of non-cases as cases will bias results towards overestimating risks.

<u>Diagnostic bias - screening</u>: In situations where screening programs are in place, there is often a bias towards over-reporting for several reasons:

This is not factor in these studies as they were not screening studies

<u>Recall Bias</u>: Recall bias is most often associated with case-control studies, where persons with and without disease are asked about their past exposure. However, the use of medical records to compile work histories, for example, has the potential to introduce such a bias. The reasons for this is that persons with a particular problem are more likely to be asked about certain factors. Recall bias could operate in the case of interviews of persons with lung cancer to obtain smoking histories. This might also affect the information obtained concerning past work histories. There is also a differential bias in that the next of kin may know less than the individual case.

CONFOUNDING:-

Confounding represents an association between variables where each may play a role in the causation of the disease in the population under study. For example, a study of lung cancer in a population containing a higher proportion of smokers than the population with which comparison is made, may provide a result which is in fact a reflection of the difference in smoking habits between the two populations. If smoking is not considered, another parameter which distinguishes the populations may be reported as responsible for the difference in the lung cancer risk.

Confounding seriously weakens a study in terms of its value for reaching conclusions about causation.

Smoking was only taken into account in one of the GTC studies.

EXPOSURE-RESPONSE:-

One would expect the strength of a genuine association between an agent and disease to increase as the level of exposure to the agent increases or as the duration of exposure to that agent increases and/or both. For example a standardized mortality ratio which increases with increasing exposure of workers is consistent with a causal association.

The absence of a clear exposure-response relationship suggests that a causal relationship does not exist or that the method chosen to express exposure is not appropriate.

This is extremely important and is a major strength of the study by Delzell et al [1995]

SPECIFICITY OF RISK TO DISEASE SUB-GROUPS:-

If a particular cancer cell type is much more common in workers exposed to a certain chemical (having taken account of confounders) while other cell types are equally common in the exposed and non-exposed workers, this would be a strong argument in favour of causality.

The identification of a risk specific to a particular disease subgroup can be strong evidence in support of causality.

SPECIFICITY OF RISK TO EXPOSURE SUB-CATEGORIES:-

If only workers working in the plant during the years that a specific chemical was produced, had an increased risk, this would add considerably to the weight of evidence concerning causality.

The concentration of an increased risk in space and/or time adds strength to arguments for causality.

STRENGTH OF ASSOCIATION:-

The basic rule is the higher the observed increase in risk, the less likely that other factors explain the excess, unless the other factors are themselves likely to produce a similar high risk (See Confounding).

Relative risks of less than 2.0 may be readily explicable by some unperceived bias or confounding factor, while those above 5.0 are less likely to be so explained.

While it is not impossible for an agent to pose a low risk and be the causal agent, conclusions that associations are causal when relative risks are low at high exposure may be in error.

CONSISTENCY:-

Both internal and external consistency are important. Do the increases in risk occur in the categories of exposure when expected and in all the subgroups where expected? Do the results of the various studies provide the same or consistent results?

The same or similar results in several studies adds support to arguments concerning causality. However, the strength of each study should be individually taken into account. Often negative studies do not get published, so several studies suggesting a weak association, do not automatically lead to acceptance of causation.

In this case, as far as is known all the studies undertaken have been examined. On the other hand, there is only one study in New York State in which exposure was adequately examined. Importantly, there has been considerable consistency between the results of various researchers. All have reported an overall increased risk of lung cancer. All have found a greater risk in the shorter term employees and several have found no or a low risk in millers and a high risk in miners.

TEMPORAL RELATION OF RISK TO EXPOSURE:-

A long latent interval appears to be involved in lung cancer and longer still in mesothelioma, with some tumours occurring more than 30 years after first exposure. It is obvious that the disease should occur after the exposure and also should occur after what might be a feasible latency interval in the case of cancers.

Evidence that an excess of disease occurs outside the anticipated/suspected latency period, suggests that more work is needed on latency prior to reaching any conclusions about causal links between exposure and disease.

In his study, Gamble made the point that his data are consistent with a smoking etiology and the latency for smoking puts the exposures prior to joining the GTC plant.

LACK OF ALTERNATIVE EXPLANATIONS:-

This is not a very sound basis for establishing causality, although sometimes invoked. In practice, the knowledge base changes, and associations that we cannot explain today may have explanations in the future.

BIOLOGICAL PLAUSIBILITY:-

Biological plausibility is very important.

Even though an association appears strong, the absence of biological plausibility detracts from causality

The mechanism by which asbestos induces lung cancer and mesothelioma is still under discussion. In this case, it seem likely that the only "regulatory definition fibres" are likely to be talc fibres, transitional fibres and cleavage fragments. Whether these cause lung cancer or mesothelioma in animal and in vitro system studies is important. Epidemiological studies of other workers exposed to non-talc cleavage fragments have not suggested increased lung cancer risks.

CHANCE:-

While strength of association takes chance into consideration, it must be appreciated that associations described epidemiologically are often statistical in nature. There is always a chance that an association which is not real will appear to be statistically significant by chance. It is easy to understand that at the 5% level of probability, 1 in 20 associations tested statistically could be significant as the result of chance alone). This means that when multiple tests are done, results must be interpreted taking "chance" into consideration.

The independence of tests must also be considered. When the same cases are included in more than one test, the results for the second test are not independent of the first.

The weight given to statistical tests or confidence intervals in considering causality must be evaluated in the context of all available data.

POWER:-

The power of a study refers to its ability to detect an effect. Often this is reported to be solely a factor of population size. This is not totally the case, as the reliability of exposure classification, exposure levels and other factors can determine whether an effect is detected.

The power of a study is important when considering whether a risk exists or not.

EXPERIMENTAL CRITERIA: Sometimes, there are no human data. When this is the case, there is the need to consider whether the evidence from animal experiments is adequate to conclude causation in animals and then to consider whether it is justified to extrapolate from the animals species studied to humans. Such extrapolation demands that differences between species are fully understood, this includes differences in metabolism, biological protective mechanisms, genetics particle biopersistence and other factors which may modify or render extrapolation invalid.